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Schaumberg, K., Brosof, L. C., Lloyd, E. C., Yilmaz, Z., Bulik, C. M., Zerwas, S. C., & Micali, N. (2020). Prospective associations between childhood neuropsychological profiles and adolescent eating disorders. *European Eating Disorders Review*, *28*(2), 156-169. https://doi.org/10.1002/erv.2721

Peer reviewed version

Link to published version (if available):
10.1002/erv.2721

Link to publication record in Explore Bristol Research
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Prospective Associations between Childhood Neuropsychological Profiles and Adolescent Eating Disorders

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RUNNING HEAD: Neuropsychological predictors of eating pathology

Abstract

Objective: Cross-sectional associations between eating disorders (EDs) and deficits in neuropsychological functioning have been well documented; however, limited research has examined whether neuropsychological functioning is prospectively associated with EDs. The current study investigated prospective associations between neuropsychological functioning in childhood (ages 8 and 10) and ED behaviors and disorders in adolescence (at ages 14, 16, and 18 years) in a population-based sample. **Method:** Participants (N = 4803) were children enrolled in the Avon Longitudinal Study of Parents and Children (ALSPAC), a population-based, prospective study of women and their children. Regression methods tested associations between facets of neuropsychological functioning (attention, working memory, inhibition) and eating disorder symptoms and diagnoses. **Results:** Better scores on working memory tasks in childhood were associated with decreased risk of fasting but increased risk of excessive exercise during adolescence. Better inhibitory control was associated with decreased risk for disordered eating at age 14, and attentional difficulties were associated with increased risk for binge eating disorder during adolescence among boys, but not girls. **Conclusions:** Neuropsychological functioning may enhance risk for disordered eating behaviors in specific ways. Overall, effect sizes were small, and results did not support global associations between neuropsychological differences and ED risk in this sample.

Keywords: ALSPAC, eating disorders, working memory, attention

Highlights

- We examined associations between childhood neuropsychological functioning and adolescent eating disorder diagnoses and symptoms
- Better scores on working memory tasks in childhood related to decreased risk of fasting but increased risk of excessive exercise during adolescence
- Results did not support global associations between childhood neuropsychological functioning and eating disorder risk

Neurocognitive functioning may be altered among individuals with eating disorders (EDs), with differences noted across several neurocognitive domains, including attention, working memory, and inhibitory control (Smith, Mason, Johnson, Lavender, & Wonderlich, 2018). A recent systematic review noted that patterns of neurocognitive functioning in EDs vary across ED subtypes and highlighted the paucity of prospective research designs that can effectively answer whether neurocognitive differences exist prior to the onset of EDs or are a consequence these disorders (Smith et al., 2018). Temporality has been explored by comparing individuals in recovery from EDs with healthy controls with mixed findings (Lozano-Serra, Andrés-Perpiña, Lázaro-García, & Castro-Fornieles, 2014; Tchanturia et al., 2012), though it is also possible that alterations in neuropsychological functioning among recovered individuals represent a 'scar' of the illness. As EDs often present in adolescence, identifying childhood risk factors, such as neurocognitive profiles, that predict later eating concerns can assist in developing a more accurate mechanistic understanding of EDs as well as aid in early identification of at-risk youth.

Effective attentional control requires individuals to orient towards and process specific information, and individuals with EDs often show impaired functioning in this domain. For instance, patients with bulimia nervosa (BN) and binge-eating disorder (BED) show elevated comorbidity with attention-deficit hyperactivity disorder (ADHD)(Fernandez-Aranda et al., 2013), and often report difficulties with attention. Individuals with anorexia nervosa (AN), on the other hand, demonstrate difficulty integrating input at a broad, global level and instead perseverate on situational details, a phenomenon termed "weak central coherence" (Lang, Lopez, Stahl, Tchanturia, & Treasure, 2014)

Working memory functioning refers to the ability to hold and work with information in mind and use information to guide behavior (Baddeley, 1992). Studies that compare working memory between those with EDs and healthy controls have yielded mixed results (Smith et al., 2018). Some studies have evaluated working memory over the course of treatment in individuals with AN and reported improvements in working memory following recovery from the illness, and deficits in working memory may be associated with longer duration of illness (Brooks, Funk, Young, & Schioth, 2017). In contrast to research that demonstrates deficits in working memory among those with EDs, other findings indicate relatively *high* scores on measures of both intelligence and working memory in this population (Lopez, Stahl, & Tchanturia, 2010). Further, recent evidence highlights a positive genetic correlation between AN and measures of educational attainment, suggesting that the same risk markers that predispose risk to AN also increase the likelihood of a greater number of years of education, a measure which is generally positively associated with working memory (Duncan et al., 2017).

In addition to attention and working memory, a third neurocognitive domain that often demonstrates differences among individuals with EDs is inhibitory control—a range of processes that reflect the ability to suppress or interrupt behavioral response (Bari & Robbins, 2013). The majority of studies investigating inhibitory control in EDs have focused on differences between healthy individuals and those who binge eat, with the hypothesis that individuals who exhibit binge eating behavior are more likely to also have deficits in inhibitory control (Wierenga et al., 2014). Findings have generally supported this notion, with evidence that deficits in inhibiting responses that have not been initiated might be particularly marked as compared to the ability to stop an already initiated behavior (Smith et al., 2018). Some have hypothesized that individuals

who exhibit restrictive-spectrum behaviors may show *enhanced* inhibitory control (Wierenga et al., 2014), though this possibility has not been thoroughly examined.

A limitation to the majority of existing research on neuropsychological risk for eating disorders is that neuropsychological deficits may be a consequence of illness processes, including malnutrition and comorbid psychopathology (e.g., depression, anxiety), i.e., 'scars' of the ED. Little research has included a prospective design to investigate neurocognitive functioning prior to the onset of disease. Moreover, reliance on clinical samples focuses examination of the role of neurocognitive processes for individuals who seek treatment, which are a minority of ED sufferers in the community (Solmi, Hatch, Hotopf, Treasure, & Micali, 2014; Swanson, Crow, Le Grange, Swendsen, & Merikangas, 2011). In addition to the need for prospective, epidemiological research, evaluation of neurocognitive risk at the level of behavioral and cognitive symptoms can buttress understanding of ED risk. From a conceptual perspective, specific neurocognitive deficits are expected to give rise to specific ED symptoms (e.g., deficits in inhibitory control are hypothesized to give rise to binge eating), which often cross diagnostic boundaries, whereas existing research has often relied on diagnostic-level distinctions. Evaluation of how neurocognitive risk relates to particular ED behaviors (e.g., fasting, purging, binge eating, excessive exercise) could inform our understanding of the etiology of disorders and support new therapeutics. Further, little research has evaluated relationships between neuropsychological function and cognitive eating disorder symptoms, such as fear of weight gain, thin-ideal internalization, and dietary restraint, which exist on a continuum in the community and can represent intermediate ED phenotypes.

The Avon Longitudinal Study of Parents and Children (ALSPAC) is a relatively large cohort of children with rich phenotype information, including neuropsychological functioning

during childhood and subsequent ED symptoms and diagnoses in adolescence. A next step in characterizing neuropsychological risk for EDs across development includes examining whether childhood neuropsychological profiles, prior to the age of ED onset, indeed relate to subsequent ED symptoms and diagnoses during adolescence. The current study capitalizes on epidemiological data on domains of neurocognitive functioning, including inhibitory control, attention, and working memory, in middle childhood (ages 8 and 10), and examines whether functioning in these domains relates to ED symptoms and disorders throughout adolescence (ages 14, 16, and 18). Given that measures of childhood neuropsychiatric functioning were collected on this cohort as they aged, as part of the routine data collection, and independent of any associations with eating disorders, we are limited in our investigation by those measures that were collected. In accordance with previous studies, we expected that attentional deficits would relate to increased likelihood of a range of ED behaviors, while difficulties in inhibitory control would specifically relate to binge eating and purging behaviors.

Method

Participants

Participants were children enrolled in the Avon Longitudinal Study of Parents and Children (ALSPAC) study, an epidemiological, longitudinal study of mothers and their children (Boyd et al., 2013; Fraser et al., 2013; Golding, Pembrey, Jones, & ALSPAC, 2001). Women who were expecting to deliver a child between 1st April 1991 and 31st December 1992 in Avon, UK were invited to take part in the study. Interested expectant mothers provided informed and written consent. Children (n = 14,062) from 14,451 pregnancies were enrolled; at one year, 13,988 children were alive. At seven years, 713 additional children were enrolled in the cohort (Boyd et al., 2013). The study website contains details of all the data that is available through a

fully searchable data dictionary and variable search tool: http://www.bris.ac.uk/alspac/researchers/our-data/. Ethical approval for this study was obtained from the ALSPAC Ethics and Law Committee and the Local Research Ethics Committees. Informed consent for the use of data collected via questionnaires and clinics was obtained from participants following the recommendations of the ALSPAC Ethics and Law Committee at the time. Detailed phenotypic, exposure, and socio-demographic data were collected via self- and maternal-report, face-to-face assessments (Boyd, et al., 2013). With regards to data relevant to the current study, 7,488 children attended face-to-face data collection waves at age 8 and 7,563 at age 10. 5,938 children completed eating disorder questionnaire measures at age 14, 5,131 at age 16, and 3,372 at age 18.

Measures

ED Behaviors

All ED behaviors were self-reported and were measured at 14, 16, and 18 years of age. For each ED behavior, questions inquired about the previous year and were adapted from the Youth Risk Behavior Surveillance System (YRBSS) questionnaire (Kann et al., 1996), which were validated in an epidemiological study of youth (Field, Taylor, Celio, & Colditz, 2004). Binge eating was defined as eating a very large amount of food and feeling out of control during these episodes. Purging was defined as making oneself sick or using laxatives to lose weight or avoid gaining weight. Fasting was assessed with the question "During the past year, how often did you fast (not eat for at least half a day) to lose weight or avoid gaining weight?" with endorsement of at least once per month coded as 'present'.

Participants rated their frequency of binge eating, purging, and fasting, which were then dichotomized as present or absent. Compulsive exercise was defined as exercise for weight loss

or to avoid weight gain. At age 14, participants rated their frequency of engaging in exercise to lose weight (0 = never, 1 = sometimes, 2 = frequently), which was dichotomized as present (1 or 2) or absent (0). At ages 16 and 18, exercise was assessed by a question asking whether participants exercised for weight loss and experienced guilt due to missing an exercise session. At these time points, compulsive exercise was defined as reporting exercise for weight loss with *sometimes* or *often* experiencing guilt if they missed an exercise session. In addition to a rating of presence or absence of ED behaviors at each assessment point, a lifetime variable was calculated for each behavior, such that individuals who endorsed engaging in that behavior at *any* age (14, 16, or 18) were coded as Yes (present = 1) or No (absent = 0).

ED and Related Cognitions

All of the following measures were self-reported at 14 years of age.

Body dissatisfaction was assessed using the Body Dissatisfaction Scale of the Satisfaction and Dissatisfaction With Body Parts Scale (Berscheid, Walster, & Bohrnstedt, 1973). This scale asks individuals to rate their satisfaction with nine body parts on a 5-point scale, from 'extremely satisfied' to 'extremely dissatisfied,' (Cronbach's $\alpha = 0.84$ in the current study). A continuous score was derived for this measure, with higher values indicating higher dissatisfaction.

Fear of weight gain was assessed through one item asking the degree to which participants have worried about gaining a little weight (0 = not at all, 1 = a little, 2 = a lot, 3 = all the time).

Pressure to lose weight was assessed through six items asking the degree to which participants feel pressure to lose weight and was adapted from the Perceived Sociocultural Pressure Scale (Stice, Nemeroff, & Shaw, 1996).

Emotional eating, external eating, and restrained eating were assessed through using 25 items of the Dutch Eating Behaviors-Questionnaire [DEBQ; (Van Strien, Frijters, Bergers, & Defares, 1986)], which were rated on a five-point Likert scale. The DEBQ raw scores are totaled into three subscale scores: Emotional Eating (eating in reaction to emotions), External Eating (eating in reaction to external cues), and Restrained Eating (cognitively attempting to limit one's caloric intake). All emotional and external eating items were used in this study; only two items of the restraint subscale were used as a measure of restraint. Higher scores on each subscale indicate greater symptomatology.

Thin ideal internalization was assessed using the Ideal-Body Stereotype Scale-Revised [IBSS-R; (Stice, Shaw, & Nemeroff, 1998; Stice, Ziemba, Margolis, & Flick, 1996)]. Questions were gender-specific; girls were asked five questions (Cronbach α=0.56) and boys six questions (Cronbach α=0.71; (Calzo, Austin, & Micali, 2018).

ED Diagnoses

Eating disorder diagnoses (AN, BN, BED, and purging disorder [PD]) were derived using questionnaire data from the YRBSS from adolescents using DSM-5 diagnostic criteria (American Psychiatric Association, 2013; Micali et al., 2015). Body mass index (BMI) was an objective measure collected at face-to-face assessment (median ages 13.8, 15.5, and 17.8 years) and was included as a diagnostic criterion for AN. Underweight was determined using age, gender, and BMI-specific cutoffs (based on UK reference data;(Cole, Flegal, Nicholls, & Jackson, 2007) corresponding to World Health Organization (WHO) grade 1 thinness.

Parental report of AN symptoms was also used at ages 14 and 16 when formulating AN diagnoses as prior research has shown that parental report often aids in the diagnosis of AN in adolescents due to under-reporting of AN symptoms (House, Eisler, Simic, & Micali, 2008). In

addition to threshold eating disorder diagnoses, we also identified youth with disordered eating cognitions and behaviors. This category included individuals who reported monthly binge eating, purging excessive exercise, or fasting, along with those who reported more sporadic disordered eating behaviors along with shape and weight concern at age 14, and those who reported eating disorder behaviors at any subthreshold level of severity at ages 16 and 18.

Neuropsychological Variables (predictors)

Several measures of neuropsychological functioning were collected during childhood (ages 8 and 10), as described below.

Attention

Children completed the Test of Everyday Attention for Children [TEA-Ch; (Manly et al., 2001)] at age 8. This test included multiple behavioral tasks designed to capture different aspects of attention and attentional control, including the sky search task and dual tasks described below.

Sky Search Task. In this attention task, participants are instructed to find pairs of identical crafts in the sky, which are the target items. Twenty target items are present among 108 distractor items. When participants believe they have completed the task, they mark a box in the lower left-hand corner to terminate the task. The outcome variable was time taken to complete the task. Motor control (time taken per target) was subtracted from the total in order to separate motor speed from attention.

Dual Task. The dual task is a parallel version of the Sky Search Task, in which the procedure is the same but the location of targets differs. While completing the search, participants are simultaneously presented with an auditory counting task. Participants are instructed to silently count the number of auditory tones presented while completing the visual search task. At the completion of the visual search task, participants are asked the total number

of tones presented. Similar to the Sky Search Task, the time taken per target was calculated. In addition, the number of counting items divided by the number of items attempted was calculated on the auditory counting task. Time-per-target was then divided by the proportion of correct counting items to account for poor counting performance. Finally, the Sky Search task score was subtracted from this value in order to calculate the final outcome variable.

Working Memory.

Working memory was assessed via two measures, described below:

The Freedom from Distractibility (FDI) Index. Children completed the Wechsler Intelligence Scale for Children-Third Edition (WISC-III) at their assessment visit at age 8. From this measure, the FDI index was calculated by adding the sum scores of the Arithmetic and Digit Span subtests. The FDI index is an establish index which is associated with teacher ratings of child inattention and evidences good construct validity (Anastopoulos, Spisto, & Maher, 1994). Both the digit span and arithmetic subtests of the WISC are also components of a larger "working memory index" of the WISC-IV, and involve ability to memorize information, hold it in short-term memory, and manipulate this information with reason processes.

Counting Span Working Memory Task (Case, Kurland, & Goldberg, 1982)**.** This task assesses working memory and was measured at ten years of age. Participants are presented with red and blue dots on a white screen and are instructed to count the red dots out loud. Participants are presented with sets of screens (i.e., three sets of two screens, three sets of three screens, three sets of four screens, three sets of five screens). After each set, participants are asked to recall the number of red dots on each screen within the set. A span score is calculated based on total number of correct sets (maximum of five), and a global score is calculated based on total number of correct trials (maximum of 42), independent of set. The Counting Span Task is a frequently

used measure of childhood working memory and evidences good internal consistency and convergent validity (Conway et al., 2005)

Inhibitory Control

Stop Signal Motor Inhibition Task. The Stop Signal task was assessed at ten years of age and is a measure of inhibitory control of a pre-conditioned motor response. First, children are presented with an O or an X stimulus on the screen and respond with the corresponding motor response (pressing O or X, respectively). Thirty trials are conducted to condition the appropriate motor response. From these thirty trials, a mean response time is calculated (*SS reaction time*). After these trials, 48 experimental trials are conducted (*SS experimental block*). All trials are conducted in accordance with the mean response time calculated from the 30 initial trials. During 16 randomly selected trials of the total 48 trials, participants hear a bleep and must inhibit their response. The bleep either occurs at 150 milliseconds, which is a difficult condition (SS 150 ms), or at 250 milliseconds, which is an easy condition (SS 250 ms). Outcome variables are: 1) the mean responses time from the initial 30 trials (SS reaction time), 2) the probability of inhibiting a response at the 150ms delay interval (SS 150ms), 3) the probability of inhibiting a response at the 250 ms delay interval (SS 250 ms), and 4) mean response time for non-inhibited trials during the experimental block (SS experimental). The Stop Signal Task is one of the most commonly used behavioral measures of inhibitory control and has been used to assess inhibitory control across multiple psychiatric disorders, including ADHD and schizophrenia (Lipszyc & Schachar, 2010). The Stop Signal Task evidences good discriminant validity and temporal stability (Kindlon, Mezzacappa, & Earls, 1995)

Opposite Worlds Task. The Opposite Worlds Task is part of the TEA-Ch (Manly et al., 2001), completed at age eight. This task assesses inhibitory control. Participants are given a sheet

of the digits one or two that are quasi-random. First, participants complete the "Same world" condition in which they read the digits out loud as quickly as possible as they are presented on the sheet. Then, participants complete the "Opposite world" condition, in which they must say the opposite response for each digit (i.e., "one" for two and "two" for one) as quickly as possible. The outcome variable is the total time taken to complete the task. This task evidences excellent test-retest reliability and good convergent and divergent validity.

Confounders

Child sex (in non-stratified analyses), maternal education (in all analyses) BMI at age 10 (in all analyses) were included as covariates, as these variables may associate with both neuropsychological functioning and ED symptoms and cognitions. BMI was calculated from objectively-measured height and weight at 10 years of age, evaluated at an in-person assessment. Age and sex-adjusted BMI Z-scores were used in analyses. Maternal education was used as a proxy for socioeconomic status. Maternal educational level was obtained by questionnaire at enrollment, dichotomised into: (i) ordinary-level qualifications generally obtained at age 16 years or higher; (ii) certificate of secondary school education (lowest level qualifications generally obtained at age 16 years), vocational qualification or no qualifications.

Data Analytic Plan

Participants were included if they completed at least one neuropsychological measure at age 8 or 10 along with a measure of ED behaviors at age 16 (N = 4803). Approximately half (45%) of the sample had complete data, with an additional 20% of the sample missing only eating disorder measures at age 18 (see Supplemental Table 1 for patterns of missingness).

Exclusion criteria were child being deceased, having no known address, or refusing participation in the study. For multiple births, the older twin was included and younger twin

excluded. After identifying the eligible sample, we performed a multiple imputation procedure for missing data on eating behaviors and neuropsychological variables. All neuropsychological and ED data were included in the imputation procedure and data were assumed to be missing at random. Lifetime ED diagnoses were derived *within* each imputed dataset by creating an indicator variable of whether specific behaviors and diagnoses were present at ages 14, 16, and 18, post-imputation. Simulations demonstrate that relative efficiency of 20 imputations relative to 100 imputations is >0.97 when there is <70% missing data (Graham, Olchowski, & Gilreath, 2007); thus, 20 imputations were chosen for the current analysis.

Neuropsychological variables were evaluated for sex differences. For variables that evidenced discrepancy across sex (performance on the Attention Sky Search, Dual Attention, and Opposite Worlds Tasks, along with reaction time and performance on the Stop Signal Task at 150ms delay), analyses were separated by sex to minimize confounding influence. For variables that did not exhibit gender differences (FDI; working memory global and span scores, performance on the Stop Signal Task at 250ms delay along with performance on this task in the experimental block), gender was included as a covariate. Analyses included logistic regression to evaluate associations between neuropsychological and ED behavior variables, followed by ordinary least squares regression examining the relationship between neuropsychological predictors and ED risk variables for the subset of individuals who completed these measures at age 14. As fear of weight gain was measured with a limited number of ordinal response points, we used ordinal logistic regression to estimate effects for this variable. Analyses were considered exploratory and were conducted first without correction for multiple comparison. A Bonferroni-Holm correction was then applied within each set of analyses (e.g. for each neuropsychological

measure predicting the set of ED behaviors) to determine associations that survived multiple comparison correction.

Results

ED Behaviors

A set of logistic regression procedures evaluated associations between neuropsychological and dichotomized ED behavior and diagnosis variables at ages 14, 16, and 18, along with lifetime occurrence of these behaviors (i.e. that occurred at any of these time points). Full results from these models are presented in **Table 1a-1d.**

Attention. Higher dual task decrement scores (poorer dual attention), were associated with higher levels of fasting at age 16 for girls, but not boys **(Table 1a).**

Working Memory. Higher FDI scores were associated with an increased likelihood of compulsive exercise at age 18. In contrast, higher FDI scores were associated with a decreased likelihood of lifetime engagement in fasting by age 18. **(Table 1b).**

In a similar pattern, greater Counting Span and Working Memory scores consistently related to increased likelihood of excessive exercise and decreased likelihood of fasting across adolescence, though these effects did not survive correction for multiple comparisons **(Table 1b).** Global working memory scores did significantly associate with higher levels of external eating at age 14.

Inhibition. Measures of inhibition did not evidence a relationship with ED symptoms throughout adolescence in this sample **(Tables 1a-1b**).

ED diagnoses

***Attention*.** Greater time to complete dual and opposite worlds tasks, indexing poorer attention, was associated with increased risk for BED in boys only at age 14. Time to complete

the dual task measure, indexing slowing performance and more difficulty with attention, was also related to increased risk for BED in boys at age 16 **(Table 1c).**

Working Memory. Measures of working memory demonstrated exploratory-level association with disordered eating, though these associations were no longer significant after correcting for multiple comparisons **(Table 1d),**

***Inhibition*.** Higher scores on the stop signal task (indicating better inhibition) were associated with decreased risk for disordered eating at age 14 **(Table 1d).**

ED symptoms at age 14

A subset of individuals (N = 3754) provided additional data on at ED symptoms and cognitions at age 14. We conducted additional analyses to examine the relationship between neuropsychological variables at ages 8 and 10 and ED symptoms at age 14, using maximum likelihood estimation. Results are presented in **Table 1a-1b.** Among these variables, the only findings that survived multiple comparison correction were related to external eating: higher levels of working memory and freedom from distractibility were associated with increases in external eating—eating in reaction to external cues—at age 14 **(Table 1b).**

Discussion

The current study evaluated neuropsychological functioning during middle childhood and its relation to ED symptoms during early (age 14), middle (age 16), and late (age 18) adolescence. Hypotheses regarding the specific nature of the relationship between childhood neurocognitive functioning and adolescent ED symptoms were partially supported. Working memory domains related to a *greater* likelihood of excessive exercise behavior at age 18 and with higher levels of external eating— eating in reaction to external cues—at age 14. In contrast, these measures were more likely to inversely relate to fasting, indicating that individuals with lower working memory

abilities at age 10 were more likely to report not eating for at least half a day for weight loss purposes during adolescence. The contrasting findings related to excessive exercise and fasting behavior indicate that different profiles of neuropsychological risk may be associated with specific ED behaviors. In addition, the similar pattern of findings across two measures of working memory (counting span and FDI) underline the consistency of this particular finding in this sample.

The fact that measures of working memory in childhood differentially related to excessive exercise and fasting behavior at later time points indicates that neurocognitive risk may not just be disorder-specific, but instead may be behavior-specific. Exercise for weight control, for instance, requires a level of planning and execution that may rely on a higher level of attentional and working memory capacity as compared to fasting. In contrast, the association between poorer working memory and likelihood of fasting may reflect reduced capacity for dietary planning or as a compensatory behavior in response to eating-related disinhibition. Consistent with the notion that fasting is generally an unsuccessful weight control strategy in community samples (Schaumberg, Anderson, Anderson, Reilly, & Gorrell, 2016; Stice, Davis, Miller, & Marti, 2008), previous research on the ALSPAC sample suggests that fasting behavior related to likelihood of children being overweight or obese, and that this behavior was likely in those belonging to a class of adolescents with symptoms resembling BN and/or purging disorder (Micali et al., 2017). Better working memory was also associated with increased risk of external eating at age 14, which points to the need for future work to delineate whether working memory dysfunction may result in increased attention to external food cues or permit successful dietary restriction, and whether this relationship shifts across development. As working memory is a brief objective neuropsychological measure which demonstrated a range of associations with

multiple ED behaviors and cognitions in the current study, investigation of this question is both relevant and feasible for eating disorder research.

In contrast with previous work, we did not find consistent relations between inhibition difficulties and binge eating behavior (Kittel, Brauhardt, & Hilbert, 2015; Smith et al., 2018; Wierenga et al., 2014), though slower performance on attentionally demanding tasks did relate to increased likelihood of binge eating for boys during early to mid-adolescence. More successful inhibition, assessed by performance on the stop signal task, also related to likelihood of disordered eating at age 14, supporting the notion that greater capacity for inhibition may be recruited in some capacity to attempt restriction of intake.

Overall, results supported more associations between neuropsychological functioning and ED behaviors than ED cognitions. The observed pattern may relate to ED behaviors indicating more severe pathology, as compared to ED cognitions. In contrast, these tasks were more consistently associated with disordered eating than to full-threshold diagnoses, though this may result from these analyses being better powered than analyses with full-threshold disorders. In addition, the measurement of eating disorder cognitions can only be captured by self-report. It may be that unlike behaviors, awareness and thus, report of eating disorder cognitions can vary greatly across individuals. In clinical practice, it is not uncommon for patients to deny cognitions or concerns at an earlier stage in their illness, that they may endorse later.

While some findings emerged supporting specific links between neuropsychological profiles and eating-related risk, effect sizes were small and results did not indicate sweeping risk. This pattern would, at first glance, contrast with existing literature that demonstrates consistent differences between those with eating disorders and healthy controls on several domains of neuropsychological functioning (Smith et al., 2018); however, there are several reasonable

explanations for these contrasting findings. First, measures used in the current study captured several facets of neurocognitive functioning, but were unable to capture some specific elements of neurocognitive functioning that demonstrate the have shown to be consistently associated cross-sectionally with Anorexia Nervosa [set-shifting (Roberts, Tchanturia, Stahl, Southgate, & Treasure, 2007; Wu et al., 2014); weak central coherence(Lang et al., 2014)]. As the ALSPAC study is not a prospective examination of only eating disorder development, we were restricted to the available datasets.

Second, it is possible that individuals included in clinical samples are not reflective of the broader population of individuals who experience eating disorder symptoms, as only a limited subset of individuals with ED in the population access healthcare (Solmi et al., 2014; Swanson et al., 2011). Altered neurocognitive functioning may thus be more pronounced among populations with increased severity, including those presenting to higher level treatment settings. Further, while measurement of neurocognitive functioning in childhood improves the likelihood that these measures are not influenced by eating disorder processes, it is possible that neurocognitive functioning may change over time and would show differential patterns of risk when assessed more proximally to eating pathology onset. Finally, it is possible that more marked alterations in neurocognitive functioning represent a consequence of rather than a risk factor for the development of eating pathology, or that defining recovery in terms of physical recovery (e.g. weight restoration) and behavioral abstinence when evaluating 'recovered' samples does not capture a fully-recovered population (Bardone-Cone et al., 2010).

Limitations and Considerations

The current study builds on previous cross-sectional research investigating neurocognitive correlates of EDs by examining prospective risk for specific ED symptoms in an epidemiological

cohort at three time points during adolescence. Obtaining multiple neurocognitive measures prior to the age of risk for ED onset provides compelling evidence that differences in neurocognitive performance among individuals with EDs are not purely due to ED onset or sequalae of the disorders.

While age 10 is prior to age of highest risk for ED, it cannot be ruled out that very low levels of ED symptomatology could have existed in the sample at age 10, though the magnitude of this potential effect would have minimal impact on the current results. It is also relevant that neuropsychological data were only available for a portion of the cohort. While we attempted to preserve data at age 18 through the use of multiple imputation procedures, we decided that children must have at least one neuropsychological and one ED measure to compute meaningful imputation. As such, not all participants are represented. In addition, we experienced reduced power to detect associations between neurocognitive function and some eating disorder outcomes in boys due to low levels of certain ED diagnoses and as reflected by larger standard error estimates on these outcomes. Further, participants in this sample are primarily white and all from a specific area of the UK, and it is possible that patterns of risk may differ across cultures.

Conclusions

The current study is the first epidemiological investigation of whether premorbid neuropsychological functioning during childhood prospectively relates to risk for eating disorder symptoms and diagnoses. Clinically, the current findings suggest that neuropsychological alterations may not be particularly useful to flag eating disorder risk at the population level, though it is possible that different patterns may emerge in smaller pools of otherwise high-risk groups. Given existing research connecting specific cognitive profiles, including social cognition

and attention, with eating disorder status and severity (Oldershaw et al, 2011; Smith et al. 2018), additional research at a population level is necessary to determine which facets of cognition may predispose eating pathology, and at what point during development cognitive differences may emerge. As more epidemiological data with deep phenotyping of cognitive profiles, brain function, and eating disorder risk becomes available, analysis of a range of cognitive profiles (e.g. working memory, attentional control, theory of mind) in relation to eating disorder risk will further understanding of how eating disorders develop over time.

Acknowledgement of Funding
This manuscript was supported by grants from the National Institute of Mental Health (R21MH109917 and T32MH076694). The UK Medical Research Council and Wellcome (Grant ref: 102215/2/13/2) and the University of Bristol provide core support for ALSPAC. This publication is the work of the authors and they will serve as guarantors for the contents of this paper. A comprehensive list of grants funding is available on the ALSPAC website. This research was specifically funded by NIH 5R01MH073842.

Acknowledgements:
We are extremely grateful to all the families who took part in this study, the midwives for their help in recruiting them, and the whole ALSPAC team, which includes interviewers, computer and laboratory technicians, clerical workers, research scientists, volunteers, managers, receptionists and nurses.

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Table 1a. Neuropsychological predictors of eating disorder behaviors and symptoms, stratified by gender.

Eating Disorder Behaviors [Odds Ratios (SE)]										
	Sky Search		**Dual Task**		**Opp Worlds**		**SS Reaction Time**		**SS 150 ms Delay**	
	Boys	Girls	Boys	Girls	Boys	Girls	Boys	Girls	Boys	Girls
Binge 14	1.11(0.06)	1.00(0.07)	1.00(0.01)	1.00(0.01)	1.04(0.03)	0.99(0.02)	1.00(0.002)	1.00(0.001)	0.98(0.05)	1.03(0.04)
Binge 16	1.06(0.05)	0.97(0.04)	1.00(0.01)	1.00(0.01)	1.02(0.03)	1.01(0.01)	1.00(0.002)	1.00(0.001)	1.06(0.05)	0.99(0.02)
Binge 18	1.07(0.05)	1.04(0.05)	1.00(0.01)	1.00(0.01)	1.04(0.02)	1.01(0.01)	1.00(0.002)	1.00(0.001)	1.02(0.05)	1.01(0.03)
Binge Lifetime	1.04(0.04)	1.02(0.04)	1.00(0.01)	1.00(0.01)	1.02(0.02)	1.01(0.01)	1.00(0.002)	1.00(0.001)	1.02(0.04)	1.01(0.02)
Purge 14	0.90(0.23)	1.06(0.11)	**1.02(0.01)**	1.00(0.01)	1.07(0.05)	1.01(0.02)	1.00(0.01)	1.00(0.003)	1.03(0.12)	0.93(0.05)
Purge 16	0.86(0.17)	0.99(0.05)	0.98(0.03)	1.00(0.01)	1.00(0.05)	0.97(0.02)	1.00(0.004)	1.00(0.001)	0.97(0.07)	0.98(0.02)
Purge 18	0.83(0.16)	0.93(0.07)	1.00(0.01)	1.00(0.01)	0.95(0.06)	**0.95(0.02)**	1.00(0.004)	1.00(0.001)	0.98(0.08)	1.06(0.04)
Purge Lifetime	0.85(0.12)	0.98(0.04)	1.00(0.01)	1.00(0.01)	0.99(0.04)	0.98(0.02)	1.00(0.002)	1.00(0.001)	0.97(0.05)	1.00(0.02)
Ex 14	0.96(0.05)	0.96(0.04)	0.99(0.01)	1.00(0.01)	0.99(0.02)	**0.97(0.01)**	1.00(0.001)	1.00(0.001)	0.99(0.03)	1.01(0.02)
Ex 16	0.97(0.05)	1.00(0.04)	1.00(0.004)	1.00(0.004)	0.98(0.02)	1.00(0.01)	1.00(0.001)	1.00(0.001)	1.01(0.03)	1.03(0.02)
Ex 18	0.90(0.08)	0.94(0.05)	1.00(0.01)	1.00(0.01)	0.97(0.03)	1.00(0.01)	1.00(0.002)	1.00(0.001)	1.04(0.04)	**1.06(0.02)**
Ex Lifetime	0.95(0.04)	0.97(0.04)	0.99(0.004)	1.00(0.004)	0.97(0.02)	0.99(0.01)	1.00(0.001)	1.00(0.001)	1.00(0.02)	**1.05(0.02)**
Fast 14	0.94(0.11)	1.02(0.06)	0.99(0.02)	1.00(0.01)	1.02(0.04)	1.01(0.01)	1.00(0.003)	1.0090.001)	1.01(0.07)	0.98(0.03)
Fast 16	0.86(0.16)	1.06(0.05)	1.00(0.01)	**1.01(0.004)***	1.06(0.04)	1.00(0.01)	**0.99(0.003)**	1.00(0.001)	0.92(0.07)	1.01(0.03)
Fast 18	1.02(0.11)	1.02(0.07)	0.98(0.03)	0.99(0.01)	0.98(0.06)	0.95(0.03)	1.00(0.01)	1.00(0.002)	1.01(0.10)	1.02(0.04)
Fast Lifetime	0.96(0.08)	1.01(0.04)	1.00(0.01)	1.01(0.004)	1.02(0.03)	0.99(0.01)	1.00(0.002)	1.00(0.001)	0.96(0.05)	1.00(0.02)
Eating Disorder Symptoms at Age 14 [*B* (SE)]										
BD	0.03 (0.10)	**-0.30(0.13)**	0.02(0.01)	-0.02(0.01)	0.07(0.05)	-0.01(0.03)	0.004(0.003)	-0.001(0.003)	0.02(0.07)	0.03(0.06)
Fear Wt Gain (OR)	1.01 (0.04)	0.99(0.03)	1.00(0.004)	1.00(0.004)	1.03(0.02)	0.99(0.01)	1.00(0.001)	1.00(0.001)	1.00(0.03)	1.00(0.02)
Restraint/Dieting	0.001(0.02)	-0.01(0.03)	-0.001(0.001)	0.001(0.003)	0.01(0.01)	-0.003(0.01)	-0.001(0.00)	-0.001(0.001)	0.00(0.01)	0.02(0.01)
Lose Wt	0.007(0.02)	-0.04(0.03)	-0.001(0.002)	0.00(0.004)	0.01(0.01)	-0.01(0.01)	-0.001(0.001)	0.00(0.001)	-0.02(0.01)	0.02(0.02)
Emotional Eating	-0.05(0.07)	-0.12(0.09)	-0.004(0.01)	-0.01(0.01)	-0.02(0.03)	-0.03(0.03)	0.001(0.002)	-0.002(0.002)	-0.01(0.05)	0.00(0.05)
External Eating	0.05(0.06)	-0.09(0.05)	0.002(0.01)	-0.002(0.01)	0.03(0.03)	-0.03(0.02)	-0003(0.002)	**-0.002(0.001)**	-0.001(0.03)	-0.04(0.03)
TI Internalization	-0.02(0.04)	0.02(0.04)	-0.01(0.004)	-0.003(0.01)	-0.01(0.02)	0.00(0.01)	0.002(0.001)	0.00(0.001)	**0.05(0.03)**	-0.01(0.02)

Note. Bolded coefficients reached significance at exploratory level ($p < .05$). coefficients with (*) significant after Bonferroni-Holm correction. Odds ratios and coefficients are presented with standard errors in parentheses. SS = Stop Signal. Sky Search = sky search task from the tests of everyday attention in children (TEA-Ch). Dual Task = dual task score from the TEA-Ch. Opp worlds = opposite worlds task score from the TEA-Ch. Fear Wt Gain = fear of weight gain. TI Internalization = thin-ideal internalization. OR = odds ratio. Body Mass Index and socioeconomic status entered as covariates.

Table 1b. Neuropsychological predictors of eating disorder behaviors and symptoms.

Eating Disorder Behaviors [Odds ratios (SE)]					
	Freedom From Distractibility	**Working Memory (Global)**	**Working Memory (Span)**	**SS 250ms delay**	**SS Experimental Block**
Binge 14	1.00(0.01)	1.01(0.01)	0.90(0.10)	1.01(0.03)	1.02(0.01)
Binge 16	1.01(0.01)	1.01(0.01)	1.08(0.07)	1.00(0.02)	0.99(0.01)
Binge 18	1.00(0.01)	1.00(0.01)	0.96(0.07)	0.99(0.03)	0.99(0.01)
Binge Lifetime	1.00(0.01)	1.00(0.01)	0.95(0.06)	0.99(0.02)	0.99(0.01)
Purge 14	**0.93(0.03)**	0.97(0.02)	0.83(0.20)	**0.91(0.05)**	0.98(0.02)
Purge 16	1.00(0.01)	1.01(0.01)	1.02(0.09)	0.99(0.03)	1.00(0.01)
Purge 18	1.01(0.02)	1.00(0.01)	1.06(0.11)	1.03(0.04)	1.00(0.01)
Purge Lifetime	0.99(0.01)	1.00(0.01)	1.01(0.08)	0.99(0.02)	1.00(0.01)
Ex 14	1.00(0.01)	1.00(0.01)	0.97(0.05)	1.00(0.02)	1.00(0.01)
Ex 16	1.01(0.01)	**1.02(0.01)**	**1.16(0.06)**	1.01(0.02)	1.00(0.01)
Ex 18	**1.03(0.01)***	**1.01(0.01)**	1.09(0.06)	1.03(0..02)	1.01(0.01)
Ex Lifetime	**1.02(0.01)**	1.01(0.01)	1.07(0.05)	1.01(0.02)	1.01(0.004)
Fast 14	**0.97(0.01)**	**0.98(0.01)**	**0.80(0.09)**	0.98(0.03)	1.00(0.01)
Fast 16	**0.97(0.01)**	**0.98(0.01)**	**0.83(0.09)**	0.99(0.03)	0.99(0.01)
Fast 18	**0.96(0.02)**	0.98(0.01)	0.90(0.11)	0.99(0.05)	1.00(0.01)
Fast Lifetime	**0.97(0.01)***	**0.98(0.01)**	**0.86(0.07)**	0.99(0.02)	1.00(0.01)
Eating Disorder Symptoms at Age 14 (*B [SE]*)					
BD	-0.03(0.02)	-0.03(0.02)	-0.22(0.15)	-0.01(0.05)	0.00(0.002)
Fear Wt Gain (OR)	1.00(0.01)	1.00(0.01)	0.98(0.05)	1.00(0.02)	0.99(0.004)
Restraint/Dieting	-0.01(0.004)	-0.001(0.003)	-0.01(0.03)	0.001(0.01)	0.00(0.002)
Lose Wt	-0.003(0.01)	-0.003(0.004)	-0.01(0.04)	-0.01(0.01)	0.00(0.003)
Emotional Eating	0.01(0.02)	0.002(0.01)	-0.01(0.12)	-0.01(0.04)	-0.001(0.01)
External Eating	**0.02(0.01)**	**0.02(0.01)***	**0.02(0.08)**	-0.01(0.03)	0.01(0.01)
TI Internalization	**0.02(0.01)**	**0.01(0.01)**	0.10(0.06)	0.01(0.02)	0.01(0.01)

Note. Bolded coefficients reached significance at exploratory level ($p < .05$). coefficients with (*) significant after Bonferroni-Holm correction. Odds ratios and coefficients are presented with standard errors in parentheses. SS = Stop Signal. Sky Search = sky search task from the tests of everyday attention in children (TEA-Ch). Dual Task = dual task score from the TEA-Ch. Opp worlds = opposite worlds task score from the TEA-Ch. Fear Wt Gain = fear of weight gain. TI Internalization = thin-ideal internalization. OR = odds ratio. Body Mass Index, gender, and socioeconomic status entered as covariates.

Table 1c. Neuropsychological predictors of eating disorder diagnoses, stratified by gender.

Eating Disorder Diagnoses (Odds Ratios [SE])										
	Sky Search		**Dual Task**		**Opp Worlds**		**SS Reaction Time**		**SS 150 ms Delay**	
	Boys	Girls	Boys	Girls	Boys	Girls	Boys	Girls	Boys	Girls
BED 14	**1.39 (0.09)***	1.05(0.19)	1.00(0.03)	0.99(0.03)	**1.19(0.05)***	1.02(0.02)	0.99(0.01)	1.00(0.004)	1.39(0.25)	1.01(0.11)
BED 16	**1.20 (0.05)***	0.89(0.11)	1.01(0.01)	1.01(0.01)	**1.11(0.04)**	0.98(0.04)	1.00(0.01)	**0.995(0.002)**	1.11(0.14)	0.95(0.05)
BED 18	1.08(0.06)	1.01(0.06)	1.00(0.01)	1.00(0.01)	1.06(0.03)	**1.01(0.01)**	1.00(0.003)	1.00(0.002)	0.94(0.07)	0.96(0.03)
BED Lifetime	1.09(0.05)	1.00(0.06)	1.00(0.01)	1.01(0.01)	1.05(0.03)	1.01(0.01)	1.00(0.002)	1.00(0.001)	0.96(0.06)	0.97(0.03)
PD 14	0.47(0.89)	0.99(0.20)	0.98(0.09)	0.98(0.04)	0.82(0.24)	1.02(0.02)	1.00(0.02)	1.00(0.004)	1.83(0.58)	0.94(0.09)
PD 16	0.80(0.34)	0.95(0.11)	0.95(0.06)	0.99(0.02)	0.98(0.11)	0.96(0.04)	1.00(0.01)	1.00(0.002)	0.96(0.15)	0.95(0.05)
PD 18	0.77(0.33)	0.94(0.12)	0.99(0.03)	1.00(0.02)	0.96(0.10)	**0.91(0.04)**	1.00(0.01)	1.00(0.003)	0.89(0.14)	1.11(0.07)
PD Lifetime	0.78(0.24)	0.93(0.09)	0.98(0.03)	1.00(0.01)	0.96(0.08)	0.96(0.03)	1.00(0.01)	1.00(0.002)	0.94(0.10)	1.01(0.04)
BN 14	0.87(0.21)	0.90(0.13)	**1.01(0.01)**	1.01(0.01)	0.98(0.07)	0.99(0.04)	1.00(0.01)	1.00(0.003)	1.09(0.12)	0.98(0.06)
BN 16	0.91(0.13)	1.01(0.06)	1.00(0.01)	0.99(0.01)	0.96(0.05)	1.00(0.02)	1.00(0.003)	1.00(0.001)	1.11(0.08)	0.97(0.03)
BN 18	0.77(0.44)	1.07(0.10)	0.97(0.05)	1.00(0.02)	0.96(0.11)	1.00(0.03)	1.00(0.01)	1.00(0.003)	1.19(0.21)	1.00(0.07)
BN Lifetime	0.89(0.11)	1.02(0.05)	1.00(0.01)	1.00(0.01)	0.96(0.04)	1.00(0.01)	1.00(0.003)	1.00(0.001)	1.10(0.07)	0.98(0.03)
AN 14	0.77(0.17)	0.94(0.09)	0.98(0.03)	1.01(0.01)	0.92(0.07)	0.99(0.03)	1.00(0.004)	1.00(0.002)	1.18(0.11)	1.03(0.04)
AN 16	0.74(0.18)	0.86(0.11)	1.00(0.02)	1.01(0.01)	1.06(0.05)	0.96(0.04)	1.00(0.004)	1.00(0.002)	1.00(0.08)	1.05(0.05)
AN 18	0.96(0.16)	0.90(0.11)	0.97(0.03)	0.98(0.02)	1.02(0.06)	0.95(0.05)	1.00(0.01)	1.00(0.003)	0.97(0.09)	1.00(0.06)
AN Lifetime	0.88(0.11)	0.89(0.08)	0.99(0.01)	1.01(0.01)	1.01(0.04)	0.97(0.03)	1.00(0.003)	1.00(0.001)	1.04(0.06)	1.05(0.04)
DE 14	1.02(0.05)	1.00(0.04)	1.00(0.01)	1.00(0.01)	1.01(0.02)	1.00(0.01)	1.00(0.002)	1.00(0.001)	**0.94(0.03)**	**0.96(0.02)**
DE 16	0.96(0.04)	0.98(0.03)	1.00(0.004)	1.00(0.004)	1.01(0.02)	1.00(0.01)	1.00(0.001)	1.00(0.001)	1.01(0.02)	1.02(0.02)
DE 18	0.95(0.29)	0.93(0.13)	0.98(0.04)	0.99(0.02)	0.99(0.10)	0.93(0.06)	1.00(0.01)	1.00(0.002)	0.96(0.16)	1.01(0.05)
DE Lifetime	0.98(0.04)	0.99(0.03)	1.00(0.004)	1.00(0.004)	1.01(0.02)	1.00(0.01)	1.00(0.001)	1.00(0.001)	0.98(0.02)	1.01(0.02)

Note. Bolded coefficients reached significance at exploratory level ($p < .05$). coefficients with (*) significant after Bonferroni-Holm correction. AN – anorexia nervosa, BN – bulimia nervosa, BED – binge-eating disorder, PD – purging disorder, DE – disordered eating. SS = Stop Signal. Sky Search = sky search task from the tests of everyday attention in children (TEA-Ch). Dual Task = dual task score from the TEA-Ch. Opp worlds = opposite worlds task score from the TEA-Ch. Fear Wt Gain = fear of weight gain. TI Internalization = thin-ideal internalization. Body Mass Index and socioeconomic status entered as covariates.

Table 1d. Neuropsychological predictors of eating disorder diagnoses

Eating Disorder Diagnoses (Odds ratios[SE]]					
	Freedom From Distractibility	**Working Memory (Global)**	**Working Memory (Span)**	**SS 250ms delay**	**SS Experimental Block**
BED 14	0.99(0.05)	0.96(0.04)	0.64(0.30)	1.07(0.12)	1.05(0.04)
BED 16	1.04(0.02)	1.03(0.02)	1.22(0.19)	0.97(0.05)	1.00(0.01)
BED 18	0.99(0.02)	1.00(0.01)	0.99(0.10)	0.95(0.04)	0.98(0.01)
BED Lifetime	1.00(0.01)	1.00(0.01)	1.01(0.09)	0.96(0.03)	0.99(0.01)
PD 14	0.92(0.05)	0.94(0.05)	0.85(0.34)	0.97(0.10)	0.99).03)
PD 16	0.98(0.03)	1.01(0.02)	0.97(01.7)	0.94(0.05)	1.00(0.02)
PD 18	0.96(0.03)	0.97(0.02)	0.83(0.18)	1.03(0.08)	1.02(0.02)
PD Lifetime	0.97(0.02)	0.99(0.02)	0.93(0.13)	0.99(0.05)	1.00(0.01)
BN 14	0.99(0.03)	0.98(0.02)	0.74(0.18)	0.95(0.05)	1.00(0.02)
BN 16	0.99(0.02)	1.00(0.01)	1.03(0.10)	0.96(0.03)	0.99(0.01)
BN 18	0.99(0.03)	1.00(0.02)	1.16(0.20)	0.98(0.07)	0.98(0.02)
BN Lifetime	1.00(0.01)	1.00(0.01)	0.99(0.09)	0.95(0.03)	0.99(0.01)
AN 14	1.01(0.02)	1.01(0.02)	1.15(0.14)	1.08(0.05)	1.01(0.01)
AN 16	1.01(0.02)	0.99(0.02)	0.94(0.15)	1.04(0.06)	0.99(0.01)
AN 18	1.00(0.03)	0.98(0.02)	0.78(0.20)	1.02(0.08)	1.00(0.02)
AN Lifetime	1.01(0.02)	0.99(0.01)	0.97(0.11)	1.07(0.04)	1.00(0.01)
DE 14	1.00(0.01)	1.01(0.01)	1.08(0.07)	**0.94(0.02)***	1.00(0.01)
DE 16	1.00(0.01)	0.99(0.01)	1.06(0.05)	1.02(0.02)	1.00(0.004)
DE 18	0.96(0.03)	0.98(0.02)	0.91(0.19)	0.92(0.06)	1.03(0.02)
DE Lifetime	1.00(0.01)	**1.01(0.01)**	1.08(0.05)	0.99(0.01)	1.00(0.004)

Note. Bolded coefficients reached significance at exploratory level ($p < .05$). coefficients with (*) significant after Bonferroni-holm correction. AN – anorexia nervosa, BN – bulimia nervosa, BED – binge eating disorder, PD – purging disorder, DE – disordered eating. SS = stop signal task. Body Mass Index, gender, and socioeconomic status entered as covariates.